

SHORT REPORT

Acalculous Gangrenous Cholecystitis Coexisting with a Mycotic Suprarenal Aortic Aneurysm

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Mycotic suprarenal aortic aneurysms are rare, and most often caused by bacteraemic spread. Acalculous cholecystitis is usually associated with major surgery and trauma. An association between acalculous gangrenous cholecystitis and a mycotic suprarenal aortic aneurysm has not been described. We present a case of a perforated gangrenous cholecystitis with an adjacent suprarenal abdominal aortic mycotic aneurysm, presenting a challenging surgical case.

Introduction

Mycotic suprarenal aortic aneurysms are uncommon, and most often caused by bacteraemic spread. Acalculous gangrenous cholecystitis is associated with a variety of clinical states, including major surgery and trauma. However, an association between both pathologies has not been previously described in the literature. We present a case of a perforated gangrenous cholecystitis with an adjacent suprarenal abdominal aortic mycotic aneurysm, presenting a challenging surgical case.

Case Report

A 62 year old man was admitted to Accident and Emergency unwell with a 4 week history of increasing abdominal and back pain, constipation and 1 year of weight loss and anorexia. His past medical history consisted of hypertension. He had recently been taking nonsteroidal anti-inflammatory medications for back pain, but no other medications. He did not smoke, nor drink alcohol.

On examination he appeared cachectic. He was tachycardic with a regular heart rate of 110, and a blood pressure of 100/60. His chest was clear on

auscultation, and heart sounds dual. He was afebrile. His abdomen was tender with some guarding over the epigastrium and right upper quadrant. He had an audible abdominal bruit. Of the lower limb pulses, only the femorals were palpable on both sides.

Laboratory investigations revealed a white cell count of 54,000/mL, and a neutrophilia. He was anaemic, his haemoglobin being 9.1 g/dL. He had abnormal liver function tests with a bilirubin of 38 μ mol/L, alkaline phosphatase of 412 IU/L, alanine transaminase of 44 IU/L, and a gamma guanine transaminase of 104 IU/L. Albumin was low at 19 g/L. He had renal failure with a urea of 19 mmol/L, creatinine of 305 μ mol/L and potassium of 5.4 mmol/L. C-reactive protein was 239 mg/L. Arterial gas sampling demonstrated a pH of 7.23, bicarbonate 22.2 mmol/L, and a base deficit of 5.2 mmol/L.

He was stabilised and blood cultures sent prior to intravenous cefuroxime and metronidazole administration. Computerised tomography revealed a leaking suprarenal saccular abdominal aortic aneurysm as well as a collection within the gall bladder (Fig. 1). Intra-arterial digital subtraction angiography was then performed, demonstrating a right sided saccular aneurysm about 4.5 cm in diameter; a 1.5–2 cm neck was noted, lying between the coeliac axis and superior mesenteric artery (SMA) origins (Fig. 2).

Laparotomy was performed using a modified Mercedes-Benz incision. Frank peritonitis was

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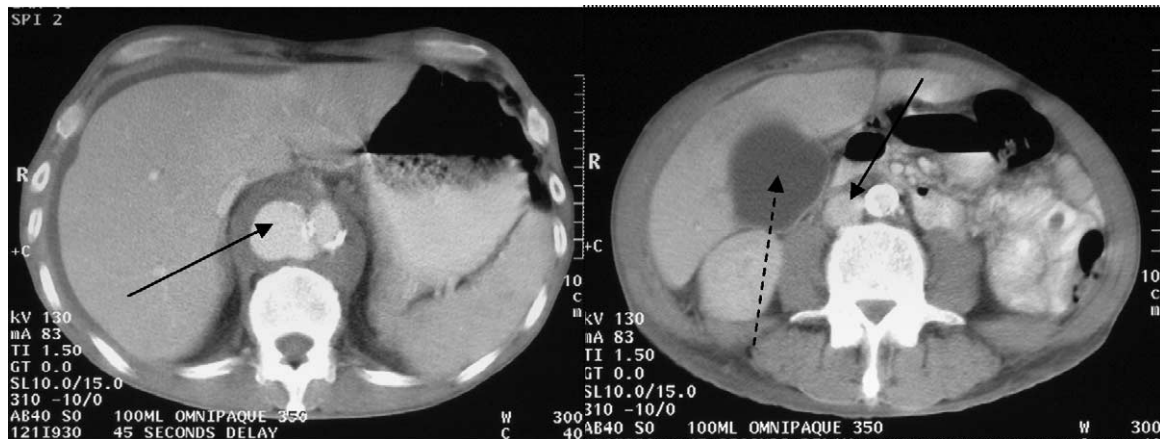


Fig. 1. Contrast computerised tomography scans of the upper abdomen revealing a leaking saccular aneurysm of the abdominal aorta (arrows), as well as a mildly distended gallbladder containing high attenuation bile, or sludge. There is also a degree of gallbladder thickening and oedema.

evident. An enlarged adherent infected perforated gallbladder was found; it was dissected and excised with ligation of the cystic artery and duct. On left medial visceral rotation multiple splenic adhesions were evident. A splenectomy was necessary following an iatrogenic tear. The aortic aneurysm was identified extending from the diaphragm to the origin of the renal arteries. Proximal clamps were applied after piercing of the diaphragm and pleura. Lack of tissue strength precluded aneurysmal neck ligation. The

coeliac axis, SMA and right renal artery origins were dissected out as a patch. A 16 mm diameter dacron graft was proximally anastomosed to aorta and the patch was anastomosed to a side hole made in the graft (**Fig. 3**). The graft was distally anastomosed directly superior to the left renal artery. Bowel and both feet were noted to be well perfused, and the abdomen was closed. The patient was transferred to recovery and then intensive care. Blood cultures taken preoperatively and in the postoperative period were consistently negative; culture of both the gallbladder and aneurysm grew *Bacteroides fragilis*. Unfortunately the patient subsequently deteriorated due to a chest infection, and died 6 weeks later of respiratory failure.



Fig. 2. Contrast intra-arterial digital subtraction angiography of the abdominal aorta showing a right sided saccular aneurysm.

Discussion

The term mycotic aneurysm was originated by Osler in the Gulstonian lectures of 1885 to designate the pathophysiology involved in the formation of mushroom shaped arterial dilation due to septic degradation of the arterial wall. The bacteria were believed to originate from infected heart valves during an episode of infective endocarditis.¹

Infected aneurysms of the abdominal aorta are relatively uncommon, accounting for only 18% of all mycotic aneurysms.² Infection of a pre-existing atherosclerotic aneurysm after bacteraemia is the most common route of infection.³ In unusual circumstances, spread from a contiguous septic process may be the cause, and it usually occurs after vascular surgery.⁴ Aneurysms of the suprarenal aorta are less frequent than those of infrarenal origin.⁵

Overall, gram-positive organisms now account for 60% of mycotic aneurysms, *Staphylococcus aureus* and

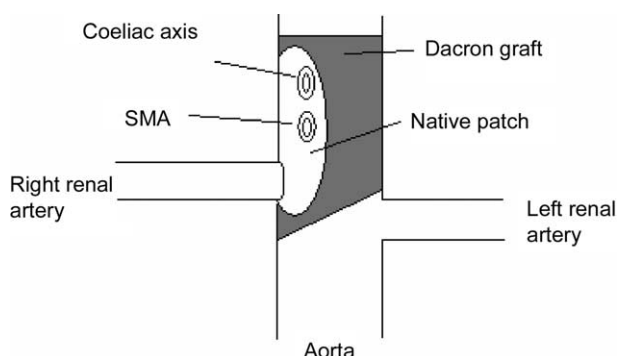


Fig. 3. Diagrammatic representation of aortic graft repair of the abdominal aortic aneurysm. Use is made of a native patch of aorta incorporating coeliac axis, superior mesenteric artery and right renal artery, which is sewn onto the graft.

Streptococcus spp. occurring in 46 and 8% of cases, respectively.⁶ The organisms implicated vary according to location. The most likely organisms involved in cases of suprarenal aneurysms are gram-negative rods, especially *Salmonella* spp.;⁷ In our case *Bacteroides fragilis* was isolated from aneurysm wall culture; this organism has been previously implicated in the literature.^{8,9}

Anaerobic bacteria are isolated in bile of 70% of patients with gangrenous cholecystitis, of which members of the *B. fragilis* group are the most common.¹⁰ We grew *B. fragilis* from the excised gallbladder. However, despite blood cultures being taken prior to antibiotic administration, these were sterile. It may be postulated then that the aneurysm infection arose from the adjacent focus. This mechanism of mycotic aneurysm formation is very unusual. Brown and associates¹¹ stated that this group constitutes less than 10% of mycotic aneurysms at all locations. Lumbar osteomyelitis, local wound infection, retroperitoneal, pancreatic and mediastinal abscesses, carcinomas and perforation of the gastrointestinal tract, infected lymph nodes and lung infections, diaphragmatic abscess and ruptured appendix have all been reported.^{2,4,12–17} However, there has been no documented case of perforated gangrenous cholecystitis to our knowledge.

We cannot be certain, however, as to whether the aneurysm was preceded by the development of cholecystitis, or *vice versa*. We know that acalculous gangrenous cholecystitis is most often associated with complicated surgery, trauma and burns,¹⁸ although diabetes, abdominal vasculitis, congestive heart failure, cholesterol embolization, and resuscitation from shock or cardiac arrest have also been associated with the condition.¹⁹ Gallbladder ischaemia and reperfusion injury, and bile stasis is thought to play a key role.^{20,21} With regard to aortic infected aneurysms,

bacterial endocarditis, arterial trauma, concurrent sepsis, and depressed host immunity have become the cardinal 'risk factors' in the development of these lesions. We could not identify a precipitant in this case with any certainty.

The definitive therapy for gangrenous cholecystitis has been cholecystectomy; percutaneous cholecystostomy is gaining acceptance as an alternative in cases of nongangrenous acute acalculous cholecystitis.²² Antibiotic treatment represents an important adjunct. The treatment of mycotic aneurysms consists of surgical resection and debridement of the infected tissue and placement of an allograft or prosthetic graft, followed by at least 6–8 weeks of antibiotics.^{16,23} But new endovascular stent-grafting techniques, followed by lifelong antibiotics, have shown some promise in the descending thoracic aorta.^{24,25} Without surgical intervention, a mycotic aortic aneurysm inevitably results in death from rupture and exsanguinations or uncontrolled sepsis; the overall reported survival rate after surgical intervention is 82 and 50% at 1 and 5 years for mycotic aortic aneurysms, with lower survival rates in aneurysmal ruptures.²⁶ In our case the aortic wall was found at operation to be too friable to successfully ligate the aneurysmal neck, and, therefore, it was decided to use a tube graft, making use of the fortunate close arrangement of coeliac axis, SMA and right renal artery to create a native patch.

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